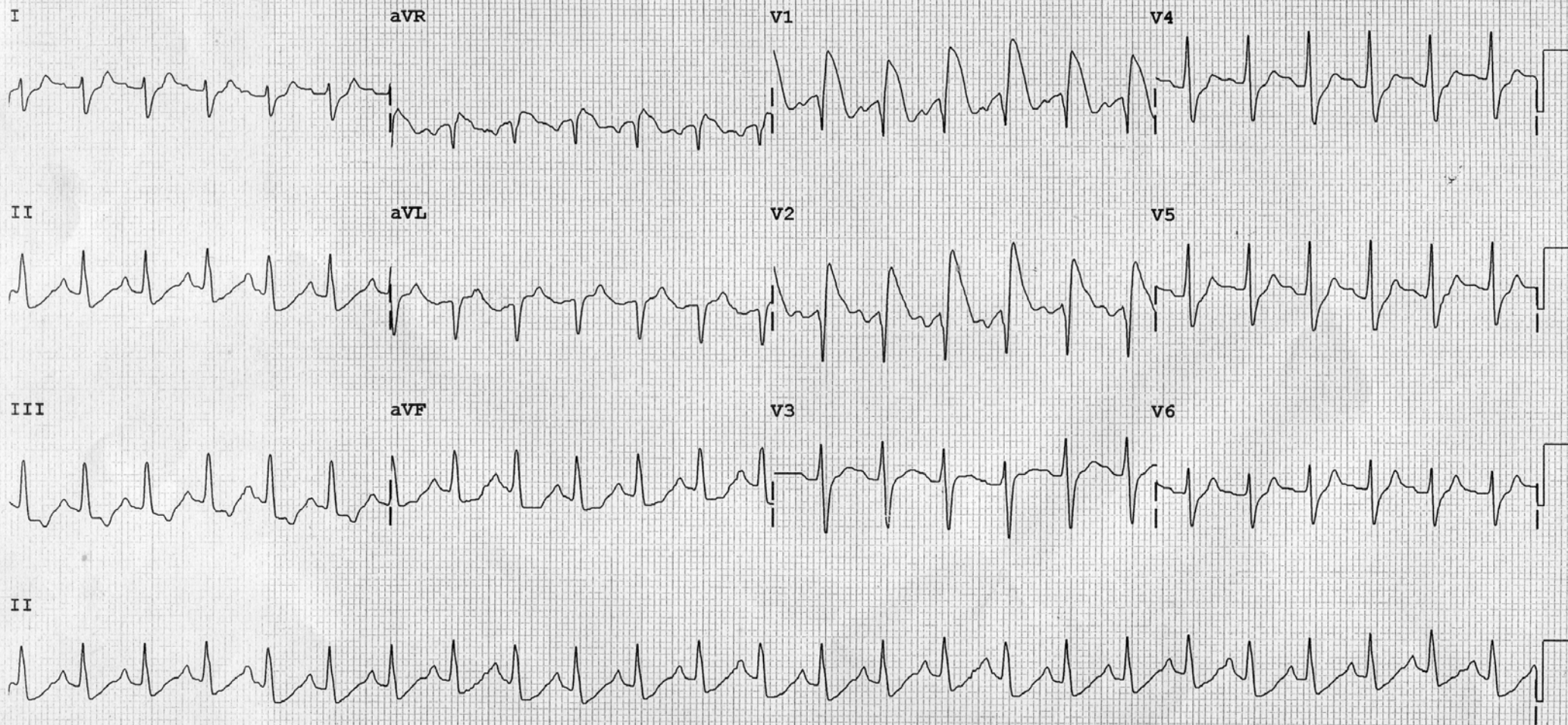


QUIZ 6th June 2018 (answers below)

1. What are the differences in paediatric ALS compared with adult ALS?
2. What are the landmarks for site of intraosseous insertion in an infant?
3. What is empagliflozin?
4. What is euDKA?
5. Describe and interpret the following ECG.



Dev: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10 mm/mV

F 50~ 0.05- 40 Hz

PH080A

P?

QUIZ answers 6th June 2018

1. What are the differences in paediatric ALS compared with adult ALS?

The paediatric ALS algorithm follows the same pattern as the adult ALS algorithm of two minutely cycles with rhythm check/pulse assessment, adrenaline every 2nd cycle, amiodarone for refractory VF/VT and working through the reversible causes. The only differences that I could think of are;

- a) The cause of cardiac arrest in paediatrics is overwhelmingly secondary to hypoxia and/or hypovolaemia, rather than a primary cardiac cause. Oxygenated breaths are started immediately and the ventilation to chest compression ratio is 15:2 rather than 30:2.*
- b) Airway opening in an infant requires a neutral position due to a soft and small airway that can kink with flexion or extension. When supine, the infant's relatively large occiput results in neck flexion, requiring slight shoulder elevation to obtain the neutral position.*
- c) Infants and children are smaller and drug doses and joules are weight based rather than flat doses.*
- d) There is almost always a parent or carer present.*

2. What are the landmarks for site of intraosseous insertion in an infant?

Proximal tibia

The tibial tuberosity can be difficult to palpate on infants so the easiest landmark is 2 fingerbreaths below the patella and then medial along the flat aspect of the tibia



Distal tibia

Place one finger directly over the medial malleolus. Move 2 fingerbreadths proximally. Palpate anterior and posterior tibial borders to confirm the flat centre aspect of the bone.



3. What is empagliflozin?

Empagliflozin is a member of the gliflozin family of drugs. Dapagliflozin and canagliflozin are also available in Australia. The gliflozins are sodium-glucose co-transporter 2 (SGLT-2) inhibitors. They are used to treat type 2 diabetes.

Glucose is freely filtered in the kidneys and then almost entirely reabsorbed by SGLT2 (90%) and SGLT1 (10%) in the tubules. This system can be saturated and when the glucose concentration exceeds the saturation point, glucose spills over into the urine. Patients with type 2 diabetes have a maladaptive increase in renal reabsorptive capacity for glucose.

Inhibition of SGLT2 lowers the saturation threshold for glucose reabsorption in the proximal tubules, thereby increasing glucose loss in the urine. Side effects can include dehydration from osmotic diuresis and increased rate of urinary tract infections due to glycosuria.

4. What is euDKA?

Diabetic ketoacidosis is a life threatening condition with the triad of hyperglycaemia, metabolic acidosis and ketosis. These patients can also present with normal, or near normal, blood glucose levels. This is referred to as "euglycaemic DKA" or "DKA with lower than anticipated glucose level" as the glucose level can still be elevated above normal. The condition is just as serious.

Causes include DKA already treated with insulin, decreased carbohydrate intake, heavy alcohol consumption, chronic liver disease, glycogen storage disorders and DKA in pregnancy can present with normal glucose levels.

The recent use of SGLT2 inhibitors is another topical and potential cause of "DKA with lower than anticipated glucose levels" although the mechanism is not clearly understood. Illness and a reduction in food intake and/or insulin dose are associated with many, but not all of these patients. SGLT2 inhibitors increase glucagon, which is ketogenic and may play a role. SGLT2 inhibitors also promote hypovolaemia from glycosuria which drives adrenaline, cortisol and glucagon which are all ketogenic.

I think the message is to be alert to ketoacidosis in these patients despite normal blood glucose level.

5. Describe and interpret the following ECG.

<i>Rate</i>	<i>150/min regular</i>
<i>P waves</i>	<i>All present and conducted. Upright in II so likely sinus in origin Look peaked but not abnormally high (<2.5mm)</i>
<i>PR interval</i>	<i>Normal</i>
<i>QRS</i>	<i>Narrow Rightward axis (between 90 – 120) RBBB pattern with large R waves in V1 and V2 Persistent S waves in V5 and V6 (RV dilatation)</i>
<i>ST segments</i>	<i>Elevation in V1, V2 and aVR Depression in V4, V5, V6, II, III and aVF</i>
<i>T Wave</i>	<i>Inversion inferiorly and V1</i>

- ➔ *Sinus tachycardia 150/min*
- ➔ *Evidence of acute right ventricular enlargement*
 - *Right axis deviation*
 - *Big R wave in V1*
 - *Big S wave in V6*
- ➔ *ST elevation in aVR, V1 and V2 associated with massive PE*
- ➔ *ST depression with TWI inferiorly can also be right heart strain*

*****Acute Pulmonary Embolus*****

*This ECG was courtesy of Marea Reading.
The patient was a 23 year old man who presented with hypotension
and hypoxia.*